GENETICS AS A TRIGGERING FACTOR FOR OBESITY

Anna Karolyne de Andrade Morais[1]
Lylian Machado Palmiere Ribeiro[2]
Augusto José de Oliveira Pereira[3]
Ruy Abdalla Soares[4]
Guilherme Rodrigues Gomes Suzana[5]
Jivago Carneiro Jaime[6]

To analyze the correlation between genetic predisposition and the development of obesity, so that predisposed individuals can prevent even more risk factors. Method: This is an integrative literature review, in which the articles were selected through the databases U. S. National Library of Medicine (Pubmed) and Virtual Health Library (VHL) databases, using the following descriptors: "genetic", "obesity" and "risk factor". The inclusion criteria were the availability of articles, those published between 2018 and 2023 and articles in English. Among the exclusion criteria were those dealing with factors other than heredity in the development of obesity. Results: Obesity is a multifactorial disease that is related to several risk factors, including genetics. Thus, it can be said that more than 250 types of genes have been found to play triggering roles in this disorder, mainly genes related to the transcription of hormones, such as leptin - known for stimulating increased energy expenditure and for its anorexigenic effects - by the LEP and LEPR genes. Also involved in the melanocortin pathway - which promotes anorexigenic and satiety effects - in this case, the POMC gene activates MC4R (involved in the aforementioned pathway), and activates the genes that code for leptin. The immune system may also be compromised, since the number of macrophages is increased (by an overexpression of S100A9 via the TLR4-NFkB pathway) proportional to the increase in adipose tissue, which stimulates the release of pro-inflammatory cytokines, which explains inflammation in obesity, This situation may also stem from mutations in the POMC gene, as it encodes the α-melanocyte stimulating hormone (α-MSH), which has an anti-inflammatory and immunomodulatory effect, and there is already a naturally increased macrophage population in the individual with the mutation. Other genes may also be mutated, such as SIM1, BDNF, and tropomyosin-related kinase B (TRKB), although they have no known action, there is a hypothesis that they act in the paraventricular nucleus of the hypothalamus, a region closely related to the hunger and satiety center, as well as being involved in brain neuroplasticity. Thus, there is a polygenic inheritance involved. Conclusion: Obesity has an important genetic component, which determines the percentage of risk of developing this disease, as exemplified by the presence of a polygenic inheritance, which should cause greater concern for individuals with a family history. However, there are also other risk factors, most of which are modifiable, such as diet, physical exercise, quitting alcohol and tobacco, among others. Thus, genetics has an important influence, however, suppression of expression and activation can be modified by the individual's lifestyle.

Keywords: Heredity; Obesity; Mutation.

[1] Student in Medicine, Evangelical University of Goiás – UniEVANGÉLICA, E-mail: annakmandrade@gmail.com
[2] Student in Medicine, Evangelical University of Goiás – UniEVANGÉLICA, E-mail: lylianpalmiere2003@hotmail.com
[3] Student in Medicine, Evangelical University of Goiás – UniEVANGÉLICA, E-mail: augustoj.op1@gmail.com
[4] Student in Medicine, Evangelical University of Goiás – UniEVANGÉLICA, E-mail: ruysoares.med@gmail.com
[5] Student in Medicine, Evangelical University of Goiás – UniEVANGÉLICA, E-mail: guirgs2017@gmail.com
[6] Teacher in Medicine, Evangelical University of Goiás – UniEVANGÉLICA, E-mail: jivagojaime@gmail.com